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# The acute effect of high-intensity interval training versus moderate-intensity continuous training on postprandial blood glucose regulation

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**Abstract**

Blood glucose control is central to the prevention and treatment of type 2 diabetes (T2D). Regular physical activity can improve glucose tolerance but many people lack the time to exercise. High-intensity interval training requires much less time commitment and results in similar or superior physiological adaptations over prolonged endurance training. However, few studies have directly compared the effects of both training strategies and a control condition on postprandial blood glucose concentration in a standardized manner. This study aims to investigate the acute effect of a single session of high-intensity interval training (HIIT) compared to a single session of moderate-intensity continuous training (CT) and a control condition (CON) on postprandial blood glucose levels. Five sedentary-recreationally active, healthy, young adults (23.6 ± 3.51 years, $VO_{2peak}$ = 33.8 (± 5.0) ml·kg$^{-1}$·min$^{-1}$) reported to the laboratory on three different occasions with a minimum wash-out period of one week to perform a single session of HIIT (10 x 60 sec of cycling efforts at 90 – 100 % $VO_{2peak}$ interspersed with 60 sec recovery cycling at 30 watts), a single session of CT (45 min of cycling at ~ 60 % $VO_{2peak}$), and a CON session in which subjects were at rest. An oral glucose tolerance test (OGTT) was performed after each trial where blood glucose levels were measured 30, 60, 90 and 120 min after ingestion of 75g glucose. Blood glucose concentration at 30, 60 and 90 min post glucose ingestion and glucose area under the curve (AUC) were reduced after HIIT compared to CON and to a greater extent compared to CT, but differences did not reach significance ($P \geq 0.05$). There is a trend that high-intensity training is superior to moderate-intensity exercise in reducing blood glucose levels. Studies with larger sample sizes are needed test this assumption.

## Introduction

Worldwide 382 million people have diabetes and numbers are estimated to rise up to 592 million by 2035 (IDF, 2013). A further 316 million people are living with impaired glucose tolerance and are at high risk for type 2 diabetes (T2D) (IDF, 2013). In the UK more than one in 17 people has diabetes (Diabetes UK, 2014), 90 % of which have T2D (HSCIC, 2013; Scottish Diabetic Survey, 2012). Spreading across all social classes and from high- to low-income countries, the disease has reached the status of a pandemic (IDF, 2013; WHO, 2015). Diabetes poses a human, financial and social burden on individuals, their families and entire countries, many of which lack the resources to cope with the disease. The human cost of diabetes is detrimental; 8.4 % of all global death among 20- to 70-year-olds is attributed to diabetes (Diabetes UK, 2014). In the U.S., 234,051 death certificates from 2010 listed diabetes as the underlying cause of death (CDC, 2014). Financially it is estimated that the economic cost of diabetes to the UK is at £13.9 billion, £13 billion of which are due to T2D, and the number is predicted to rise to £15.1 billion by 2035/6 (Diabetes UK, 2012) if a further expansion of the disease is not prevented.

T2D presents a particular challenge since the disorder can initially develop without obvious symptoms. The condition develops as a result of insulin resistance which is the principal metabolic abnormality in the metabolic syndrome (Bremer et al, 2012). Despite a greater prevalence of metabolic illness in obese individuals (Diabetes UK, 2014), 5 % - 45 % of normal-weight people also exhibit insulin resistance (Conus et al, 2007; Ruderman et al, 1998). The reality reflects this conflict: half of all people with diabetes do not even know they have it (IDF, 2014). Undiagnosed diabetes adds another £1.5 billion to healthcare in the UK (Diabetes UK, 2012); in the U.S. it was responsible for an additional USD 18 billion in one year (Zhang et al, 2009). T2D is neither a disease of the rich, nor the poor, nor is it a disease of solely the obese. Almost everyone is at risk of T2D, worldwide. Therefore it is even more important to recognise that T2D is a preventable disease.

At the centre of prevention and treatment of T2D stands the regulation of blood glucose (NICE, 2014a). Postprandial hyperglycaemia is one of the major contributors to the complications of diabetes (Ceriello, 2005) and is associated with an increased risk for cardiovascular disease resulting from diabetes (Laakso, 1999). Participation in regular physical activity can improve hyperglycaemia by enhancing glucose tolerance and blood glucose regulation (Jeng et al, 2003; Little et al, 2011; Rogers, 1989; Smutok et al, 1994; Snowling & Hopkins, 2006). Epidemiological studies suggest that physical activity can reduce the risk of T2D by 30 % – 50 % (Bassuk & Manson, 2005; Hu, 2011). Combined with weight loss and a healthy diet, moderate physical activity may even reduce the risk of developing T2D amongst those who are already at high risk by up to 60 % (Bauman, 2004). In turn, two hours per day of time spent watching television is associated with a 14 % increase in diabetes risk (Hu et al, 2003). The benefits of exercise on T2D are well established and today, exercise along with diet and weight control is considered essential for the prevention and management of diabetes (Colberg et al, 2010; NICE, 2012; Diabetes Prevention Program Research Group, 2002). The UK's national recommendations for physical activity in adults include a total of at least 150 minutes of moderate-intensity activity or 75 minutes of vigorous activity (DH, 2011; NICE, 2014b). Yet 54 % of men and 63 % of women do not meet the national recommendations for physical activity in England (Health and Social Care Information Centre, 2009). In the obese it is even less; 68 % of obese men and 81 % of obese women are inactive (Health and Social Care Information Centre, 2009). Lack of time is the most commonly stated reason not to exercise by a wide population range; for instance the working age population (Popham & Mitchell, 2005), low-income overweight and obese women (Chang et al, 2008), postpartum women (Downs & Hausenblas, 2004), individuals with diabetes (Kamiya et al, 1995), and adolescents (Tappe et al, 1989).

In recent years there has been growing evidence that training at higher intensities for a shorter duration in form of high-intensity interval training (HIIT) may result in similar or even superior physiological adaptations, including cardiovascular and metabolic changes, than achieved from continuous exercise (CT) at lower intensities (Burgomaster et al, 2005; Burgomaster et al, 2006; Gibala et al, 2006; Gibala & McGee, 2008; Jeng et al, 2003; Sandvei et al, 2012; Wisløff et al, 2007). On top of that HIIT may even be perceived as more enjoyable than moderate-intensity prolonged exercise (Bartlett et al, 2011), which may also result in increased adherence to exercise. HIIT is characterised by various combinations of short, vigorous exercise bursts interspersed with low-intensity exercise bouts or rest and is usually maintained for about 10 to 30 minutes. Energy production for these intense exercise bouts (≥ 70 % of maximal oxygen consumption ($VO_{2max}$)) lasting over ten seconds up to a few minutes relies on anaerobic pathways, specifically anaerobic glycolysis, which sources glucose from circulating blood glucose and glucose mobilised from muscle and liver glycogen in both the fed and fasted state (Marliss & Vranic, 2002). The rate of glucose disposal in the body increases more rapidly in high-intensity than in exercise at lower intensities. Catecholamine levels rise markedly which results in a seven- to eightfold increase in glucose production whilst glucose utilisation is only increased three- to fourfold (Adams, 2013). Thence glucose regulation is different in intense exercise as compared to exercise at lower intensities which depends to a greater extent on fatty acids for energy supply (Marliss & Vranic, 2002). The respiratory quotient during vigorous exercise is typically seen at about 1.0 indicating that carbohydrates and not fats are the primary fuel for exercise at high intensities (Marliss & Vranic, 2002).

These particular metabolic processes that are specific to HIIT result in physiological responses that may be beneficial for blood glucose homeostasis in healthy (Adams, 2013; Brabaj et al, 2009; Burgomaster et al, 2007; Richards et al, 2010; Sandvei et al, 2012) as well as metabolically ill individuals (Adams, 2013; Little et al, 2011; Gillen et al, 2012). For instance, six HIIT sessions repeated over two weeks reduce average 24 hour blood glucose concentration and postprandial areas under the curve for breakfast, lunch and dinner in patients with T2D (Little et al, 2011). Even a single bout of HIIT was able to reduce hyperglycaemia and postprandial hyperglycaemia in individuals with T2D (Gibala et al, 2012; Gillen et al, 2012). Long-time exposure to HIIT in a 12 week training program has further shown to improve cardiovascular fitness to a greater extent than exposure to the same frequency of steady-state exercise, and to lower blood glucose concentration after an oral glucose tolerance test (OGTT) to the same extent as prolonged low-intensity running in previously untrained men (Nybo et al, 2010).

A classic model of HIIT that has widely been used in experimental trials is the repeated Wingate test, which is an extremely demanding training protocol and requires a special cycle ergometer. It may not be applicable and particularly for metabolically ill or unhealthy individuals it may not even be safe. A more practical model has been suggested by Little et al (2010) consisting of 8 – 12 bouts of 60 sec cycling at 90 – 100 % peak power output ($VO_{2peak}$) with 75 sec of recovery between intervals. Importantly this protocol was able to provide a sufficient stimulus to improve muscle oxidative capacity and functional performance similar to that observed from the repeated Wingate tests. On top of that it was better tolerated by participants (Little et al, 2010). In 2012 Gibala, Little and colleagues modified the protocol slightly to 10 bouts of 60 sec work at a constant intensity that elicits ~ 90 % of maximal heart rate ($HR_{max).}$ This HIIT session is very time efficient as only 10 min of intense exercise are performed over a total of 20 min. Therefore the exercise protocol for the HIIT session of this study is orientated on the one suggested by Gibala, Little and colleagues (Gibala et al, 2012; Little et al, 2010).

Many studies have investigated the influence of exercise on glucose dynamics in healthy subjects and those with T2D or insulin resistance (Goodyear & Kahn, 1998; Henriksen, 2002; Thompson et al, 2001), however only few studies have tested for acute effects from HIIT and compared them to the effects from moderate-intensity CT and control on glucose homeostasis. Therefore the present study aimed to investigate the acute effect of a single HIIT session compared to a single session of moderate-intensity CT and a control (CON) condition on blood glucose regulation in a sedentary-recreationally active, healthy, young population. The outcomes of this study were blood glucose levels in response to an OGTT after the three different test situations. It was hypothesized that there would be an attenuated rise in blood glucose levels and a decrease in the glucose area under the curve (AUC) after the exercise trials compared to CON and a more pronounced effect from the HIIT session compared to the CT session.

## Methods

The study protocol was approved by the School Representative of the Science and Environment Research Ethics Committee.

### Participants

Five young, healthy, sedentary or recreationally active students, four male and one female, between the age of 18 and 35 y (mean age 23.6 ± 3.51), were recruited from Plymouth University. Mean height was 178 ± 9.5 and mean weight was 74 ± 8.7. Subjects were informed verbally as well as in written form about the purpose of the study, the experimental protocol and the potential risks before providing written, informed consent. Furthermore subjects were given information about how to prepare for the OGTT 48 hours prior to the test date.

### Pre-experimental evaluation

Prior to exercise testing anthropometric characteristics (e.g. height and mass) were measured and subjects were familiarized with the lab equipment. On their initial lab visit subjects performed an incremental cycle test to exhaustion in order to establish the maximal $O_2$ uptake ($VO_{2max}$). The $VO_{2max}$ test followed a standard protocol of 2 minute baseline cycling at a power output of 20 watts, followed by an incremental test to exhaustion with ramp rates of 30 watts·min$^{-1}$ in men and 20 watts·min$^{-1}$ in women to ensure fatigue within 8 – 12 minutes. Subsequently the work rates corresponding to 60 % and 90 % of the $VO_{2max}$ were calculated in order to normalize the training intensity for the CT and HIIT protocols, respectively.

## Experimental Protocol

A schematic of the experimental protocol is presented in Fig. 1. The study followed a non-blinded, controlled cross-over design over an 8 week period.

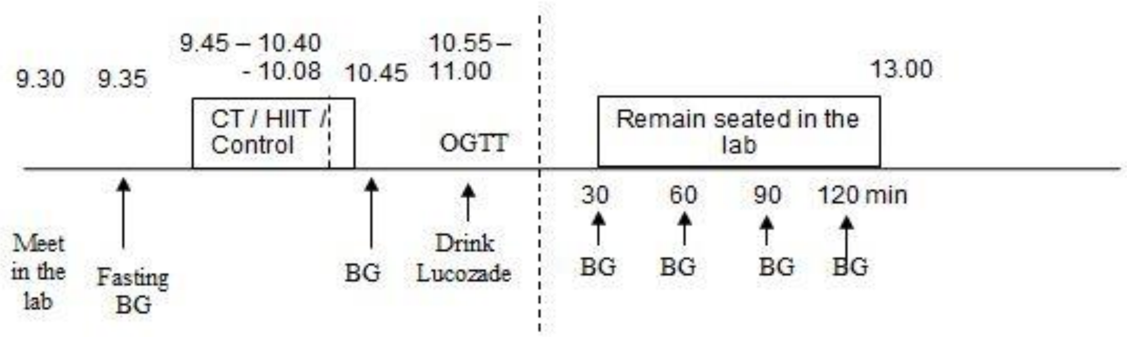


**Figure 1:** Experimental protocol for all test days. BG = Blood glucose test

Each subject served as their own control and performed both test trials which were separated by at least one week. Subjects came into the lab fasted at 9 am in the morning and fasting blood glucose was taken. On their first visit they performed the CON session in which participants were asked to remain seated in the lab. On their second visit they performed the CT and on their third visit they performed the HIIT protocol as described below. The OGTT was performed after rest or exercise at 10.45 am for all three trials.

## Exercise Intervention Protocol

All exercises were performed on the *Corvial Lode Bicycle Ergometer.* Pulmonary gas exchange during $VO_{2max}$ test, CT and HIIT was measured using the cardiopulmonary exercise testing system METALYZER® 3B and the computer software MetaSoft® Studio. The CT session consisted of 45 minutes of cycling on an electronically braked cycle ergometer at a workload designed to elicit ~ 60 % of the subject's $VO_{2max}$. Including a 3 min warm-up and a 2 min cool-down at 30 Watts, total exercise time was 50 minutes. The HIIT protocol was orientated on a practical, effective and well tolerated model suggested by Little et al (2010) and Gibala et al (2012). HIIT consisted of ten repeated bouts of 60 sec cycle exercise at 90 – 100 % $VO_{2max}$ separated by recovery periods of 60 sec cycling at low intensity (30W). Including a 3-minute warm-up and 2-minute cool-down at 30 watts, total exercise time was 25 minutes.

## Oral glucose tolerance test

OGTT was performed after consuming 75 g of glucose from 410 ml of Lucozade original following which capillary blood samples were obtained at 0, 30, 60, 90, and 120 min postprandial for measurement of blood glucose concentration. Blood samples were obtained by finger pricks and blood glucose was determined using the Accu-Check Advantage (Roche) blood glucose meter at 0, 30, 60, 90, and 120 minutes post-consumption of the Lucozade.

### Calculations and statistical analysis

Means and standard deviations were calculated for all dependent measures using Excel. The area under the curve (AUC) was calculated as the 'total area' under the curve hence including values that fall below the baseline values, so that calculations did not depend on the ever-changing baseline levels for blood glucose. This method has been suggested as a preferred method of choice for this kind of intervention by previous researchers (Potteiger et al, 2002). The formula for AUC used was:

$$AUC = (A+B)/2 \times 30 + (B + C)/2 \times 30 + (C+D)/2 \times 30 + (D +E)/2 \times 30$$

*where A = value at 0 min; B = value at 30 min; C = value at 60 min; D = value at 90 min.; E = value at 120 min*

One-way repeated measure ANOVA on Minitab was used to assess differences in blood glucose means and AUC means between all three trials. Paired T-Test on Minitab was used to assess differences in blood glucose means and AUC means between two trials. Significance was set at P-value ≤ 0.05. Effect size (ES) between AUC means for CON and CT, CON and HIIT, and CT and HIIT was measured by the mean scores / pooled standard deviation (SD). Effect sizes were classified after Cohen's term *d; small* (*d* = 0.2), *medium* (*d* = 0.5), *large* (*d* ≥ 0.8) (Cohen, 1988).

## Results

### Exercise Performance

All participants completed all the exercise tests and blood glucose tests with no complications; adherence was 100 %. None of the subjects plateaued during the $VO_{2max}$ test; hence peak oxygen uptake ($VO_{2peak}$) measures were taken. Mean $VO_{2peak}$ elicited in the ramp test was 33.8 (± 5.0) $ml \cdot kg^{-1} \cdot min^{-1}$. Mean heart rate (HR) at the end of the ramp test was 181.2 ± 6.1 beats per minute (bpm). Percentage maximal HR ($HR_{max}$) was on average 94 (± 2.8) % $HR_{max}$.

The percentage utilization of $VO_{2peak}$ during CT was 63.4 (± 5.6) %; the normalization procedure therefore successfully equated the relative training intensity for this condition (Table 1). Subjects cycled on average at 141.5 (± 12) bpm and 71.9 (± 5.7) % $HR_{max}$. On average, subjects performed under the prescribed 90 % $VO_{2peak}$ during the HIIT sprint intervals. The percentage utilization of $VO_{2peak}$ during HIIT was 80 (± 15.5) % $VO_{2peak}$ (Table 2). Subjects cycled on average at 161 (± 7.4) bpm and 82.3 (± 4.0) % $HR_{max}$.

**Table 1.** $VO_2$ values during CT compared with aimed 60% $VO_{2peak}$ values

| Subject | Target Watts (W) | 60 % $VO_{2peak}$ (ml·kg$^{-1}$·min$^{-1}$) | mean $VO_2$ (ml·kg$^{-1}$·min$^{-1}$) during CT | % $VO_{2peak}$ (ml·kg$^{-1}$·min$^{-1}$) during CT |
|---|---|---|---|---|
| 1 | 140 | 24 | 26 | 64.9 |
| 2 | 110 | 20.4 | 25 | 57.6 |
| 3 | 140* | 17.4* | 28* | /* |
| 4 | 200 | 27 | 31 | 70.4 |
| 5 | 70 | 16.8 | 17 | 60.6 |
| **Mean** | | | | 63.4 ± 5.6 |

* $VO_2$ not representable; Watt level orientated at subject 1; % $VO_{2peak}$ during CT incalculable

**Table 2.** $VO_2$ values during HIIT compared with aimed 90 % $VO_{2peak}$ values during sprints

| Subject | Target Watts (W) | 90 % $VO_{2peak}$ (ml·kg$^{-1}$·min$^{-1}$) | mean $VO_2$ (ml·kg$^{-1}$·min$^{-1}$) during HIIT | % $VO_{2peak}$ (ml·kg$^{-1}$·min$^{-1}$) during HIIT |
|---|---|---|---|---|
| 1 | 290 | 36 | 30 | 75.2 |
| 2 | 200 | 30.6 | 34 | 103.1 |
| 3 | 260* | 26.1* | 32* | /* |
| 4 | 290 | 40.5 | 31 | 70.4 |
| 5 | 160 | 25.2 | 20 | 71.4 |
| **Mean** | | | | 80 ± 15.5 |

* $VO_2$ not representable; Watt level orientated at subject 1; % $VO_{2peak}$ during HIIT incalculable

## Blood glucose responses

Table 3 and Fig. 2 show the differences between blood glucose means during CON, CT and HIIT at pre-training, post-training (0 min), and 30, 60, 90, and 120 min post glucose ingestion. ANOVA revealed no significant difference in pre-exercise blood glucose levels between CON, CT, and HIIT ($P = 0.07$). Post-exercise blood glucose levels at 0 min were also not significantly different between trials ($P = 0.46$).

**Table 3.** Blood glucose means (mmol/l) ± SD at pre-training and 0, 30, 60 90, 120 min postprandial for CON, CT, HIIT

| Trial | pre-training | 0 | 30 | 60 | 90 | 120 |
|---|---|---|---|---|---|---|
| CON | 5.2 ± 0.4 | 4.88 ± 0.3 | 8.06 ± 2.1 | 8.24 ± 3.9 | 5.64 ± 1.9 | 5.32 ± 0.7 |
| CT | 5.44 ± 0.2 | 4.66 ± 0.3 | 8.16 ± 0.9 | 6.6 ± 1.0 | 5.12 ± 0.7 | 4.52 ± 0.3 |
| HIIT | 4.94 ± 0.4 | 4.68 ± 0.2 | 7.72 ± 1.7 | 5.38 ± 1.4 | 4.78 ± 0.5 | 5.54 ± 0.6 |

There was no significant difference in blood glucose means between trials at any time ($P > 0.05$).

30 min postprandial there were no significant differences in blood glucose levels between trials ($P = 0.87$). Paired T-test revealed that differences between CON and CT ($P = 0.91$), CON and HIIT ($P = 0.79$), CT and HIIT ($P = 0.41$) were not significant. Largest difference in blood glucose concentration between all trials was reached 60 min post glucose ingestion, with no significant difference between trials ($P = 0.18$). HIIT noticeably reduced blood glucose levels compared to CON and to greater extent than CT, but results were not significant ($P = 0.12$ and $P = 0.11$, respectively). CON and CT also showed no significant differences in blood glucose levels ($P = 0.42$). 90 min postprandial there was no significant difference in glucose levels between trials ($P = 0.46$). Blood glucose levels were lowest in the HIIT trial but not significantly lower than CT ($P = 0.44$) or CON ($P = 0.41$), and differences between CON and CT were also not significant ($P = 0.40$). 120 min postprandial blood glucose levels were not significantly different between trials ($P = 0.14$). Blood glucose levels in the HIIT trial had risen after 90 min and were similarly high to after CON whilst levels after CT were lowest, with no significant difference between CON and CT ($P = 0.14$), CON and HIIT ($P = 0.22$) and CT and HIIT ($P = 0.89$).

Standard Deviation (SD) for blood glucose levels at 30, 60 and 90 min postprandial was large due to an outlier in the subject group which did not follow the usual pattern of the rest of the data (Fig 2).

**AUC during CON, CT and HIIT**

Table 4 and Fig 3 show the AUC means for CON, CT and HIIT. Highest AUC means were reached during CON (229.2 ± 256.4 mmol x min/l), CT elicited the second highest AUC mean (176.4 ± 62.4 mmol x min/l) and HIIT reached the lowest AUC mean (113.1 ± 70.8 mmol x min/l). ANOVA revealed no significant difference between AUC means ($P = 0.436$). Paired T-Test showed no significant difference between HIIT and CON ($P = 0.32$), HIIT and CT ($P = 0.01$) and CT and CON ($P = 0.65$). Effect size between CON and CT was small ($ES = 0.33$), between CON and HIIT moderate to large ($ES = 0.71$), and between CT and HIIT very large ($ES = 0.95$).

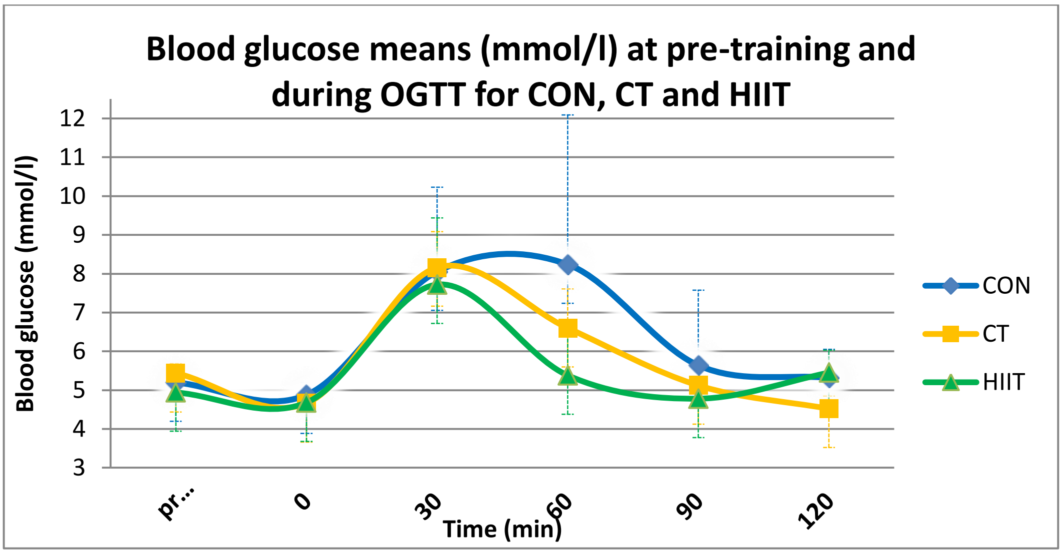


**Figure 2:** Mean blood glucose concentrations of all subjects assessed by capillary blood samples over 120 min on a non-exercise control day (CON), following a single session of 25min high-intensity interval training (HIIT) and following a single session of 50 min moderate-intensity continuous training (CT). Difference in blood glucose means between trials at any time were not significant (P > 0.05).

**Table 4.** AUC means ± SD for CON, CT and HIIT

| | CON | CT | HIIT |
|---|---|---|---|
| **AUC means** | 229.2 ± 256.4 | 176.4 ± 62.4 | 113.1 ± 70.8 |

There was no significant difference between trials (P > 0.05).

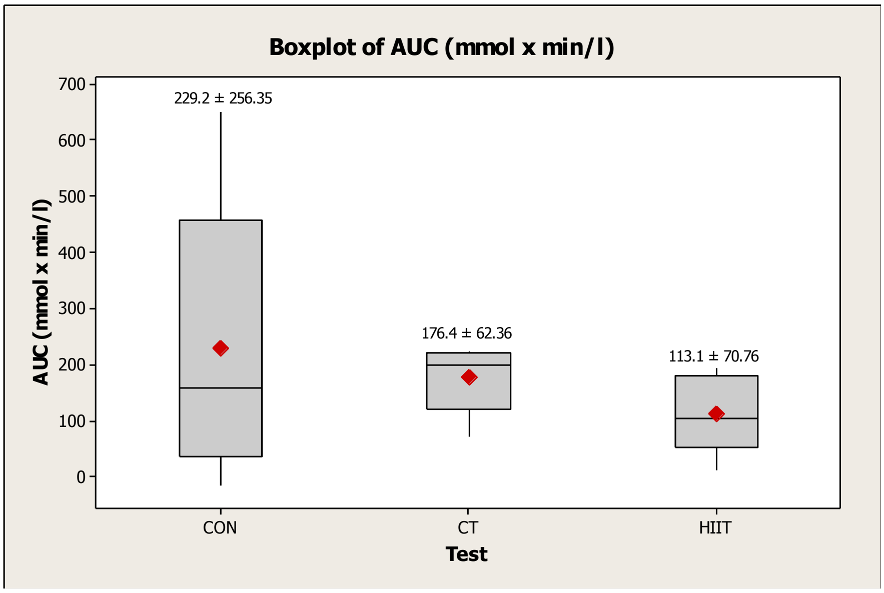


**Figure 3.** Boxplot of AUC (mmol x min/l) means ± SD for CON, CT and HIIT. There was no significant difference between AUC means (P > 0.05). Effect size was: CON vs CT: 0.33; CON vs HIIT: 0.71; CT vs HIIT: 10.66

## Discussion

Blood glucose control stands in the centre of the prevention and treatment of T2D and insulin resistance (NICE, 2014a) and due to its beneficial effects on glucose tolerance and postprandial metabolism (Jeng et al, 2003; Little et al, 2011; Rogers, 1989; Smutok et al, 1994; Snowling & Hopkins, 2006), exercise is a cornerstone for the delay and management of these metabolic conditions (Colberg et al, 2010; NICE, 2012; Diabetes Prevention Program Research Group, 2002). Yet, most people lack the time (Chang et al, 2008; Downs & Hausenblas, 2004; Kamiya et al, 1995; Popham & Mitchell, 2005; Tappe et al, 1989) to meet the national guidelines for physical activity, especially those at risk of insulin resistance and T2D (Health and Social Care Information Centre, 2009). Meanwhile, there has been growing evidence that much shorter intermittent training at high-intensities results in equal or even superior metabolic responses as seen from low- to moderate intensity prolonged exercise, including beneficial effects on hyperglycaemia, blood glucose dynamics and insulin sensitivity (Boulé et al, 2001; Burgomaster et al, 2005; Burgomaster et al, 2006; Gibala et al, 2006; Gibala & Gee, 2008; Jeng et al, 2003; Richards et al, 2010; Sandvei et al, 2012; Wisløff et al, 2007). However only few studies have compared high-intensity intermittent exercise to moderate-intensity continuous exercise and even less have also compared the results to a control condition. In this present study blood glucose levels during an OGTT were tested in five sedentary to recreationally active, healthy, young participants after 25 min of high-intensity intermittent cycling at 80 (± 15.5) % $VO_{2peak}$ (HIIT) and after 50 min of moderate-intensity continuous cycling at 63.4 (± 5.6) % $VO_{2peak}$ (CT). Results were compared to a control condition in which subjects were at rest, with a wash-out period of at least one week between all three trials.

The most prominent finding of this study was a noticeable, albeit non-significant, lower rise in blood glucose levels after a single bout of HIIT compared to a single bout of moderate-intensity CT and compared to CON, as demonstrated in Fig. 2. Largest differences in blood glucose levels between all trials were reached 60 min after glucose ingestion: average blood glucose levels were elevated to 8.24 ± 3.85 mmol/l in the control trial and lowered to 6.6 ± 1.0 mmol/l after CT and to 5.38 ± 1.4 mmol/l after HII.T However differences between HIIT and CON, HIIT and CT and CON and CT were not significant ($P = 0.12$, $P = 0.11$, and $P = 0.42$, respectively). These results are in agreement with Gillen et al (2012) who followed a similar study design to the present study and also showed a reduction in post-meal glucose concentration after an acute bout of HIIT. The same HIIT protocol as the one used in this study was applied. The study group reported that a morning session of HIIT decreased postprandial peak glucose concentration for lunch, dinner and breakfast the next morning in patients with T2D aged 62 ± 3 years. Further, 24 h glucose monitoring revealed that the sum of all three hour postprandial glucose AUC's was significantly lower following HIIT (Gillen et al, 2012). Little et al (2011) showed comparably beneficial results after repeating six sessions of the same HIIT protocol with T2D patients in a training intervention over two weeks. Unfortunately, Gillen et al and Little et al only compared these results relative to control but not relative to the effects of low- or moderate-intensity exercise. Thus, it cannot be said from these investigations whether HIIT was more effective than or as effective as CT in lowering blood glucose. However an important implication of the study by Gillen et al (2012) and Little et al (2011) is that it confirms the applicability of the HIIT protocol used in the present study to older people and individuals with T2D.

Interestingly, in the HIIT trial of this present study blood glucose concentration showed a rise again after reaching lowest levels 90 min post glucose consumption. At 120 min blood glucose levels were higher than concentrations in the CT and CON trial. This pattern may indicate that cells underwent gluconeogenesis to synthesise new glucose due to very low blood glucose levels.

The present study further showed a reduction in average AUC values after both training interventions compared to CON with a greater reduction after HIIT than after CT; though differences in AUC means did not reach significance ($P = 0.436$). However, an important finding was that effect size was large for mean AUC's between HIIT and CON (ES = 0.71) and for HIIT and CT (ES = 0.95). This shows that the effect of HIIT on blood glucose was strong whereas CT only had a small effect on blood glucose. It also indicates that the small sample size of this study may have been a major reason for the results not to reach significance. The findings are in agreement with recent results from Rynders et al (2014) who found that the glucose area under the curve during an OGTT was reduced to a significantly greater extent after a single bout of high-intensity exercise compared to a single bout of moderate-intensity exercise. However HIIT differed from this present study in that exercise was performed constantly at ~ 83 % $VO_{2peak}$ for 23.8 ± 5 min, whereas subjects of the present study performed a total of 10 min at 80 ± 15.5 % $VO_{2peak}$ interspersed with 60 sec of rest. The HIIT protocol used by Rynders et al (2014) was more intense since performance at high intensity was twice as long which may have accounted for more pronounced and significant effects. It can be speculated that if participants of the present study had not underperformed at 80 (± 15.5) % $VO_{2peak}$ but maintained the prescribed 90 % $VO_{2peak}$, perhaps 10 min performance would have been sufficient to produce significant results.

Whilst this study presents the acute effects of a single HIIT session on blood glucose regulation, it has also been shown that HIIT has a chronic effect on postprandial blood glucose concentrations. After a two week long intervention with six sessions of sprint interval training, postprandial blood glucose levels in healthy young males were still lowered three days after the last session (Brabaj et al, 2009). There is broad evidence about beneficial acute and chronic effects of HIIT on physiological markers from two week long training periods (Brabaj et al, 2009; Gibala et al, 2006; Little et al, 2010; Little et al, 2011). However less research has investigated how chronic exposure to HIIT will impact on the body in the long term. It has been shown that 15 weeks of high-intensity intermittent exercise were significantly more effective in decreasing fasting insulin levels than the same frequency of steady-state exercise (Trapp et al, 2008). Moreover a 12 week training program of intense interval running was as effective as prolonged moderate-intensity running in lowering blood glucose levels 2 hours after oral ingestion of 75g glucose compared to pre-training in previously untrained men (Nybo et al, 2010). On top of that intense interval running was superior to prolonged moderate-intensity running in improving cardiovascular fitness (Nybo et al, 2010). These results are promising considering that the intense interval running group only spent a total of 40 min per week exercising, whilst the prolonged running group spent more than triple the amount of time at 150 min per week. It has been speculated that improvements in glucose homeostasis after long-term exercise interventions may be caused by weight loss rather than exercise itself (Weinstock et al, 1998). However there was no significant change in body weight after the 12 weeks in the sprint interval running group in the study by Nybo et al (2010); therefore, reduction in blood glucose occurred independent from weight loss.

Nevertheless there has also been controversy about the safety of chronic HIIT performance particularly in individuals with impaired glucose tolerance. It has been argued that despite improving glucose homeostasis high intensity exercise may cause mechanical and oxidative damage, for example by producing high levels of free radicals (Wright et al, 2001).
After all it may be worth not thinking of acute and chronic effects as two separate mechanisms but rather as synergistic outcomes. Since acute high-intensity training improves the capacity to exercise it consequently enables longer and more intense sessions, which in turn result in more pronounced effects from a single exercise session.

The present study cannot elucidate the mechanisms that were responsible for the lowering effect of HIIT on postprandial blood glucose concentration, but understanding the biochemical processes that may underlie the effects found in this study is pivotal for further research. There are two main features that are unique to HIIT; recruitment of a large amount of muscle fibres (Gibala & McGee, 2008) and a high degree of muscle glycogen synthesis (Burgomaster et al, 2006; Praet et al, 2006). Both factors taken together imply that more muscle fibres need to replenish glycogen stores, thus causing a greater increase in subsequent muscle glucose uptake than after moderate-intensity exercise. It has been suggested that some cell signalling pathways mediating these processes may be affected by exercise and specifically to greater extent by high-intensity exercise. For instance, exercise enhances the diffusion of glucose into the skeletal muscle by regulating the translocation of glucose transporter type 4 (GLUT-4) isoform from intracellular sites to the sarcolemma (Hayashi et al, 1997; Henriksen et al, 2002; Holmes & Dohm, 2004; Hood et al, 2011; Kennedy et al, 1999; Kristiansen et al, 1996, 1997; Little et al, 2011; Richter & Hargreaves, 2013). Skeletal muscle is the main site for glucose uptake and the maintenance of a functional transport system is crucial to maintain glucose homeostasis. In turn impaired glucose transport is thought to be the main cause for hyperglycaemia (Holmes & Dohm, 2004). Insulin-dependent translocation of GLUT4 is generally impaired in patients with insulin resistance and T2D. Exercise however offers a mean to increase blood glucose uptake via a pathway that is independent of insulin. Therefore the enhancing effect of exercise on the glucose transport system is of particular interest for patients with impaired insulin function. The translocation of GLUT4 to the plasma membrane was for example demonstrated in T2D patients after a single bout of exercise (45 – 60 min at 60 – 70 % of $VO_{2max}$) (Kennedy et al, 1999). Early studies on rats were even able to show glucose transport by contracting muscle activity in the complete absence of insulin (Ploug et al, 1984).

Exploring the mechanisms involved in the translocation of GLUT4 may clarify whether high-intensity exercise has more pronounced effects on the translocation of GLUT4 than moderate-intensity exercise. Several signalling pathways are believed to play a role in contraction-induced glucose transport including calcium, protein kinase C, glycogen, adenosine, nitric oxide, and 5’ adenosine monophosphate-activated protein kinase (AMPK) (Richter et al, 2001). In addition to its stimulating effects on glucose uptake, AMPK also plays a role in insulin secretion (Salt et al, 1998). Since AMPK in skeletal muscle is activated by contraction, it has been suggested that disuse of the AMPK signalling system through sedentary lifestyle and inactivity may be a particularly potent contributor to the aetiology of T2D (Winder & Hardie, 1999). Interestingly, Wojtaszewski (2000) found that that human activation of the isoform α2-

AMPK during exercise is dependent on exercise intensity (Wojtaszewski et al, 2000). A three- to fourfold activation of α2-AMPK was observed immediately after 60 min of high-intensity exercise at 75 % $VO_{2max}$, yet no activation of α2-AMPK was found after 90 min of low-intensity exercise at ~ 50 % $VO_{2max}$ (Wojtaszewski et al, 2000). However it has to be taken into account that high-intensity training in this study was maintained constantly for a relatively long duration of 60 min. In contrast, HIIT is usually maintained for a much shorter duration. Therefore it remains unclear whether short intense exercise bouts and lower total training time as usually seen in HIIT would have the same potential to raise AMPK levels. This could be an interesting issue to investigate in future research.

Glucose uptake does not solely rely on GLUT4 translocation but also GLUT4 expression which has also shown to be increased after exercise (Richter & Hargreaves, 2013). It is tempting to speculate that due to its enhancing effects on AMPK and signalling pathways, high-intensity exercise may cause greater expression of GLUT4 than low- and moderate-intensity exercise. Indeed an increase in GLUT4 content of 119 % (Little et al, 2010) and 260 % (Hood et al, 2011) compared to baseline was found in sedentary but healthy individuals after performance of six HIIT sessions over two weeks. Moreover Little et al (2011) reported a striking ~ 369 % increase in GLUT4 content after two weeks of similar HIIT training in subjects with T2D. However these studies did not provide a comparison with exercise at lower intensities to test for potential superiority of HIIT in promoting GLUT4 expression. Kraniou et al (2006) however tested GLUT4 expression after 60 min of low-intensity exercise at ~ 39 % $VO_{2peak}$ compared to 27min of high-intensity exercise at ~ 83 % $VO_{2peak}$ and reported similar increases in GLUT4 gene expression after both trials. Despite that a superiority of HIIT could not be proven, these results are promising in that much shorter time commitment for exercise still results in similarly increased GLUT4 expression.

Increased expression and translocation of GLUT4 is not only beneficial for glucose uptake *per se,* but expression of GLUT4 also determines insulin responsiveness and plays an important role in the capacity of skeletal muscle to stimulate insulin action (Kern et al, 1990; Hollozy, 2005). Increased insulin action on the other hand enhances glucose uptake (Ryder et al, 2011) and stimulates glycogen synthesis (Calder, 1991). It has been shown that insulin sensitivity is increased up to several hours after exercise (Wasserman et al, 1989). For instance, two weeks of vigorous interval training were able to significantly improve insulin sensitivity in sedentary and recreationally active adults (Brabaj et al, 2009; Hood et al, 2011; Richards et al, 2010). The effects of HIIT on insulin action are interesting and promising for individuals with insulin resistance, and ultimately T2D patients.

The results from this present study confirm the presented hypothesis to the extent that a trend in lowered postprandial blood glucose levels and AUC after HIIT was found; however this study was unable to show significance for any result. There are several limitations to this study. A major limitation is the small sample size. Taking into account the large effect sizes in AUC means for CON and HIIT and for CT and HIIT, it is likely that sample size was the limiting factor and that results may have reached significance with a bigger sample size. Furthermore the participants were uneven in that there were 4 males but only 1 female who was also older and had less cardiorespiratory fitness than the other subjects who generally showed similar baseline data. This may have been a further hindrance for results to reach significance since it caused an outlier in the data. The subject showed a much higher rise in blood glucose levels outside of the blood glucose level range shown by the other subjects. Interestingly, if the outlier was taken out there was in fact a significant difference found in blood glucose levels between CON and HIIT at 60 min postprandial ($P = 0.04$).

Moreover there is the possibility of human error during calibration of the testing equipment and equipment error during testing. This may indeed have been the case for one of the $VO_{2max}$ tests. Subject 3, a 22 year old male, scored a $VO_{2max}$ of 24 ml/min/kg which for a 20 - 29 year old male is under the 'Very Poor' classification (Heyward, 1998). Since the subject is a healthy, active, young male who reported to participate in regular exercise, his low $VO_{2max}$ score was assumed to be false due to either human error, by faulty set up or calibration of the equipment, or due to equipment failure. Since this subject resembled much to subject 1 in anthropometric characteristics and reported activity levels, the watt level for CT and HIIT was orientated at Subject 1. In addition the subject was verbally asked to rate its perceived exertion on a scale from 1 to 10; where 1 corresponds to 'very light' and 10 to 'maximum effort'. The aim was a rating 6 during CT and 9 during the sprints of HIIT. Yet, the realised % $VO_{2peak}$ during CT and HIIT could not be calculated for this subject as shown by the dash in Table1 and Table 2.

Finally, the order of the test sessions was not randomised which is a further limitation to this study.

## Conclusion

The findings of the present study are inconclusive as the results were not significant. However, there was a noteworthy trend in that a single bout of HIIT reduced postprandial blood glucose levels to a greater extent than a single bout of moderate-intensity CT in healthy young adults. The lack in significance may be explained by the small sample size of the study. It would be interesting to examine the same experimental protocol with a larger sample size. Furthermore this study investigated the acute effect of HIIT on blood glucose concentrations. Importantly, the long-term effects of the proposed training strategy in both healthy and metabolically ill individuals have to be examined in order to draw the right conclusions and make recommendations. There is also still controversy about the exact exercise intensity and minimal time commitment needed to result in desired physiological outcomes from HIIT. Clarification about these details would be of large interest to enable the design of individual exercise protocols that can be implemented in the prevention and therapy of diseases related to metabolic dysfunctions such as T2D. Establishing the underlying physiological mechanisms responsible for metabolic improvements from HIIT would help to determine the precise exercise stimulus needed to result in particular health benefits. Despite some recent promising suggestions the full extent of the underlying biomechanics remains yet uncertain and requires more research.
HIIT offers an effective, well tolerated and safe strategy to improve metabolic and cardiovascular function. On top of that HIIT is time-efficient and potentially more enjoyable than traditional prolonged endurance exercise, thus it may result in sustained adherence. Due to these unique features, HIIT represents an interesting strategy for the management of metabolic conditions on a public as well as individual level. Research about HIIT may make a substantial contribution to tackle the detrimental economic and social cost of inactivity related diseases on society.

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